

Simultaneous ossification of the posterior longitudinal ligament and ossification of the ligamentum flavum causing upper thoracic myelopathy in DISH: case report and literature review

Qunfeng Guo · Bin Ni · Jun Yang · Zhuangchen Zhu · Jian Yang

Received: 12 January 2010 / Revised: 26 June 2010 / Accepted: 18 July 2010 / Published online: 10 August 2010
© Springer-Verlag 2010

Abstract A rare case of a 44-year-old Chinese male with diffuse idiopathic skeletal hyperostosis (DISH) and simultaneous ossification of the posterior longitudinal ligament (OPLL) and ossification of the ligamentum flavum (OLF) at T1–2 causing thoracic myelopathy is reported herein. Posterior decompression without extirpating the OPLL was performed at T1–2. Postoperatively, symptoms were greatly improved, with remaining hyperreflexia and Grade 4/5 muscle strength in the lower extremities. The Japanese Orthopedic Association score improved from 5 preoperatively to 9 at final follow-up. The presence of a cyst due to leakage of cerebrospinal fluid was confirmed by MRI at day 27, but it resolved after conservative management. The clinical manifestation of DISH, the relationship among DISH, OPLL, and OLF, and management of thoracic myelopathy due to OPLL and OLF were reviewed.

Keywords Diffuse idiopathic skeletal hyperostosis · Ossification of the posterior longitudinal ligament · Ossification of the ligamentum flavum · Upper thoracic · Myelopathy

Introduction

Diffuse idiopathic skeletal hyperostosis (DISH), ossification of the posterior longitudinal ligament (OPLL), and ossification of the ligamentum flavum (OLF) at the upper thoracic spine rarely occur simultaneously. In this paper,

the clinical manifestation, radiological evaluation, and management of such a case with thoracic myelopathy at T1–2 are reviewed.

Case

A 44-year-old Chinese male who had experienced nuchal pain and left lower extremity soreness for 2 years complained of a 15-day history of gait embarrassment and worsening of the left lower extremity ache. Neurological examination revealed bilateral lower extremity hypermyotonia and hyperreflexia, which were more significant on the right side. Manual muscle testing revealed Grade 4/5 muscle strength in the lower extremities. X-ray and CT scan documented ossification of the anterior longitudinal ligaments (OALL) at C2–7, OPLL at C3–7 and T1–2, and OLF at T1–2 (Fig. 1a–c). MRI demonstrated severe spinal cord compression due to OPLL and OLF at T1–2, with intramedullar high intensity on T2-weighted images (Fig. 2a, b).

The posterior thoracic canal was decompressed via a laminectomy at T1–2. Intraoperatively, we found that the left OLF, which was strongly adhered to the dura mater, had fused with the ventral OPLL. Considering the high risk of spinal cord injury with further extirpation of the OPLL, we ceased the decompression procedure when the dural sac pulsation resumed.

Postoperatively, the weakness in the left lower extremity worsened to Grade 3/5 muscle strength, but it gradually recovered to the preoperative level after Prednisolone was administered. The patient was discharged from the hospital at day 6. However, he presented again at day 27 because of a nuchal swelling. MRI demonstrated leakage of cerebrospinal fluid (CSF) (Fig. 3). The

Q. Guo · B. Ni (✉) · J. Yang · Z. Zhu · J. Yang
Department of Orthopedics, Changzheng Hospital, The Second
Military Medical University, 415 Fengyang Road, Huangpu
District, Shanghai 200003, People's Republic of China
e-mail: nibin99@sohu.com

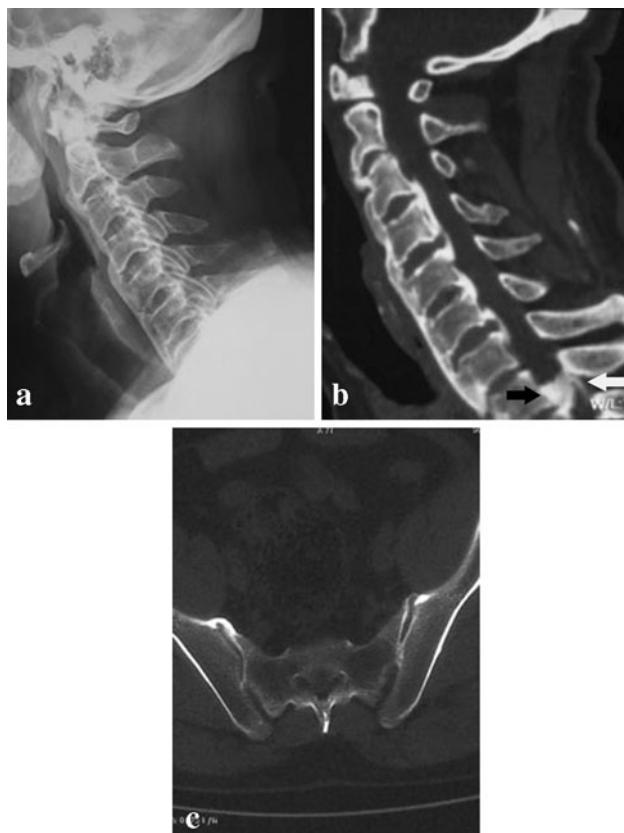


Fig. 1 X-ray and CT scan documented OALL at C2–C7, OPLL at C3–C7 and T1–2, and OLF at T1–2. **a** OALL at C2–C7, **b** OPLL (black arrow) and OLF (white arrow) at T1–2, **c** no sacroiliac erosion

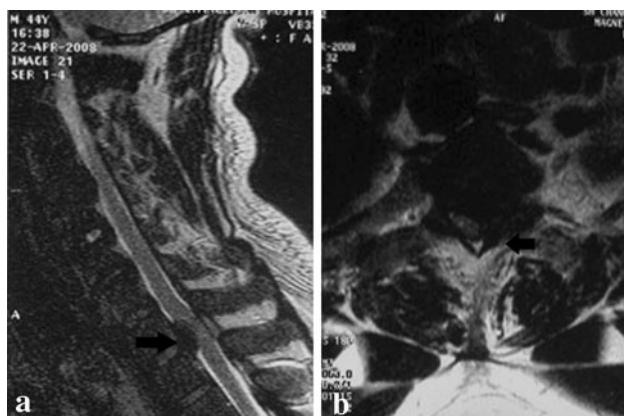


Fig. 2 MRI showed OPLL and left OLF with severe spinal cord compression and intramedullary high intensity on T2-weighted images at T1-2. **a** OPLL (black arrow), **b** OLF (black arrow)

subcutaneous CSF was aspirated with syringes after local skin disinfection, and local compression was performed. The swelling decreased and only a small amount of epidural CSF was detected on MRI at month 6 (Fig. 4a, b). From then on, the patient did not undergo MRI



Fig. 3 Postoperative MRI at day 27 revealed leakage of cerebrospinal fluid (black arrows)

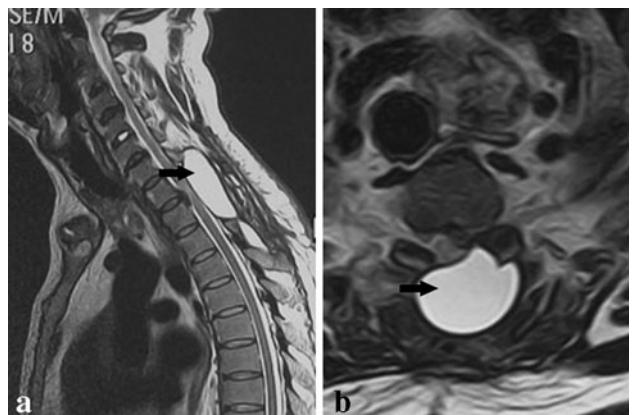


Fig. 4 Postoperative MRI at month 6 showed residual cerebrospinal fluid at the dorsal area of the dural sac (black arrows). **a** Sagittal plane, **b** axial plane

examination due to absence of nuchal pain and local swelling. At final follow-up, 18 months after surgery, the patient's symptoms had greatly improved and he was without nuchal pain and lower extremity soreness and weakness. The Japanese Orthopedic Association (JOA) score improved from 5 preoperatively to 9 at final follow-up (Table 1). Despite the remaining hyperreflexia and Grade 4/5 muscle strength in the lower extremities, he was satisfied with the results and declined further anterior decompression.

Discussion

Clinical manifestation

DISH, also known as Forestier's disease, is an idiopathic rheumatological abnormality in which exuberant ossification occurs along ligaments throughout the body. It is most

Table 1 Pre-/postoperative Japanese Orthopaedic Association (JOA) scores for thoracic myelopathy

Function	Preoperative	Postoperative
Motor function		
Lower extremity: gait	1	3
0: Unable to walk		
1: Needs aid on flat ground		
2: Needs aid on stairs		
3: Needs no aid on stairs but is unstable		
4: Normal		
Sensory function		
Lower extremity	0	1
0: Complete or apparent sensory loss or severe paresthesia		
1: Mild sensory loss or paresthesia		
2: Normal		
Trunk	1	2
0: Complete or apparent sensory loss or severe paresthesia		
1: Mild sensory loss or paresthesia		
2: Normal		
Bladder function	3	3
0: Complete retention or complete incontinence		
1: Incomplete retention, incomplete incontinence, or straining		
2: Frequency or hesitation		
3: Normal		
Total score (normal, 11)	5	9

characterized by ligamentous ossification of the anterolateral side of the spine, sparing the disc and joint space [1–4]. Although typically asymptomatic, DISH has been associated with neurological problems [5–20] (Table 2), of which the reported rate is 4% [21].

DISH alone can cause spinal cord compression, mostly in the upper cervical spine. The common pathology includes retroodontoid masses [6–8], atlantoaxial pseudoarthrosis [9], basilar impression combined with dens hypertrophy [10], and atlantoaxial subluxation [13, 14]. Fusion of the subaxial cervical spine associated with DISH was reported to result in greater stress to the ligaments of the still-mobile occipitoatlantoaxial segment, causing partial tears of the ligaments. Cycles of injury and reparative process or reactive hypertrophy contributed to the formation of this compressive pathology and subsequent neurological deterioration [6, 8, 14, 22]. In such cases, surgical management is helpful in stopping neurological deterioration. Several procedures, such as transoral decompression combined with posterior fusion [6, 8, 10, 13], decompression by the lateral approach with occipitocervical fusion [7], and single posterior decompression without fusion [9, 14], have produced good clinical outcomes. In our opinion, posterior fusion may be mandatory because single decompression may aggravate the instability of the upper cervical spine, thereby leading to neurological deterioration [8].

Neurological problems due to OPLL or OLF associated with DISH also have been reported. In DISH, most reported cases of myelopathy due to OPLL occurred in the subaxial cervical spine [11, 15, 16], and extensive posterior decompression was the common method for treating such lesions [11, 15]. However, in some cases of severe stenosis due to huge OPLL, single posterior laminectomy may not be sufficient to decompress the spinal cord [11, 15]. Additional anterior decompression may be required, which is technically demanding due to OALL. In the previous studies, OLF associated with DISH played an important role in the development of neurological deficits in the thoracic or lumbar spine [18–20]. Because OLF often occurred at the still-mobile levels, above and/or below which the spine was ankylosed [18–20], we postulated that the pathomechanism might be similar to that in occipitooatlantoaxial lesions. Chronic and continuous stress at the nonunion level contributed to hypertrophy or OLF. In these cases, posterior decompression and stabilization could achieve excellent outcomes [18–20].

In our study, a rare case of upper thoracic myelopathy due to simultaneous OPLL and OLF at T1–2 in DISH was studied. X-ray and CT scan documented OALL at C2–7, OPLL at C3–7 and T1–2, and OLF at T1–2, with C7–T1 remaining mobile. Therefore, the OLF was presumed to be stress-induced ossification due to concentrated stresses between the ankylosed subaxial cervical spine and the fixed

Table 2 Cases with neurological problems in DISH

Series [Ref. No.]	Case no.	Case Sites	Pathology	Surgery	Complications	Neurological improvement
Jun et al. [6]	1	Upper cervical spine	Retroodontoid pseudotumor; OALL from C2 downward	Transoral decompression and dorsal atlantoaxial fusion		Remarkable
Storch et al. [7]	1	Upper cervical spine	Retroodontoid mass; OALL at C2–3 and at C4–7 with occipitocervical fusion	Decompression by lateral approach with occipitocervical fusion		Remarkable
Patel et al. [8]	5	Upper cervical spine	Retroodontoid masses; OALL from C3 to C7 in all cases	4 with transoral decompression and occipitocervical fusion; 1 with posterior decompression and occipitocervical fusion	Pneumonia; graft dislodging, needing revision and Occ3 fusion; retrolisthesis of C1 on C2, needing posterior C1–2 fusion	Remarkable; 1 died at postoperative week 3 due to pneumonia
Goto et al. [9]	1	Upper cervical spine		Posterior decompression		Remarkable
Pascal-Moussellard et al. [10]	1	Upper cervical spine and C3–4	Pseudoarthrosis with large osteophytes between the posterior tubercle of C1 and the spinous process of C2			
Chiba et al. [13]	1	Upper cervical spine	Basilar impression; dens hypertrophy; hypertrophy of the ligamenta flava at C3–4; OALL at C2–3 and C4–7	Transoral decompression, C3–C4 fenestration and occipitocervical fusion		Remarkable
Kawabori et al. [14]	1	Upper cervical spine	Atlantoaxial subluxation; OPLL at C2–4; OALL at C2–T2	Posterior decompression from the occiput to C6 and occipitooaxial fusion	Spinous process fracture of C2	Remarkable
Griffiths et al. [11]	1	Subaxial cervical spine	OPLL at C3–7, with severe spinal stenosis at C3 and C5	Laminectomy from C2 to C7		Slight
Pouchot et al. [12]	1	Subaxial cervical spine	OPLL at C2–7; congenital spinal stenosis	Conservative treatments		Unchanged
Epstein et al. [15]	2	Subaxial cervical spine	OPLL	Laminectomy		Remarkable
Chacko et al. [5]	3	Subaxial cervical spine	OPLL	Multilevel cervical oblique corpectomy	Homer's syndrome; C5 radiculopathy	unchanged in 1
Razmi et al. [16]	1	Subaxial cervical spine	OPLL at C2–4 combined with C3/4 disc prolapse; minor trauma; osteophytes at C5–6	Conservative management		Remarkable
Alenghat et al. [17]	1	Subaxial cervical spine	Osteophytes at C5–6	Anterior decompression		Remarkable
Reisner et al. [18]	1	Thoracic spine	Facet joint hyperostosis and OLF at T8–T9; OALL at T11–L5	Decompressive laminectomy from T8–to T12		Remarkable
Wilson and Jaspan [19]	1	Thoracic spine	OLF at T11; OALL at T8–11 and at L2–5	Thoracic laminectomy		Remarkable
Chi et al. [20]	1	Lumbar spine	OLF at L2–L3; OALL at C2–L2 and at L3–S1	Posterior lumbar interbody fusion (PLIF)	Transient complete paraplegia	Remarkable

thoracic spine. Posterior decompressive laminectomy without extirpating the OPLL yielded good results.

Associations of DISH, OPLL, and OLF

Several previous reports discussed the association of DISH, OPLL, and OLF [23–26]. Resnick et al. [25] described 4 patients with co-existing DISH and cervical OPLL, and they found OPLL in 50% of 74 additional patients with DISH after reviewing their cervical spine radiographs. Ehara et al. [24] found that DISH was present in 27 out of 109 cases of OPLL (25%) and in 4 out of 18 cases of OLF (21%), and 3 out of 18 patients with OLF had OPLL (16%). Moreover, they found a clear trend in the distribution of these disorders. DISH predominantly occurred in the thoracolumbar spine, OPLL in the cervical spine (93% in cervical spine, 5.5% in thoracic spine, and 4.5% in lumbar spine), and OLF in the lower thoracic spine (67% in lower thoracic spine, 6% in upper thoracic spine, 17% in cervical spine, and 11% in lumbar spine). In another study, Ono et al. [23] found that the prevalence of thoracic OPLL was 0.6% and that three times as many women as men were affected; in contrast, cervical OPLL occurred predominantly in men. Because thoracic OPLL is frequently associated with DISH in men but not in women, they suggested that sex may be important in determining whether OPLL co-exists with DISH and that OPLL should not be regarded merely as a form of DISH. In addition, they found that thoracic OPLL can be associated with OLF (14/39, 36% in women; 4/7, 57% in men). Moreover, with cryptorrhea, the thoracic OPLL could increase in thickness, resulting in radiculomyopathy, and it could co-exist with cervical and/or lumbar OPLL and DISH. However, they did not discuss the possible relationships among these three disorders.

It remains unclear whether DISH, OPLL, and OLF are genetically related. COL6A1 is a gene that encodes the $\alpha 1$ chain of Type VI collagen, an extracellular matrix protein, and might provide a scaffold for osteoblastic or preosteoblastic cells or chondrocytes that subsequently proceed to membranous or endochondral ossification [27]. Because it's a susceptibility to the occurrence of DISH and OPLL in the Japanese population, COL6A1 was thought to be responsible for the hyperostotic state that led to ectopic bone formation in the spinal ligament [28]. However, no data are available to confirm this premise. On the other hand, as common clinical and metabolic features of OPLL and DISH can suggest their common aetiopathogenesis, a genotyping study on the COL 11 A2 gene was done in a group of 60 Czech patients with DISH. But results of analysis of intron 6 (-4) polymorphisms in the COL 11 A2 gene did not agree with data from Japanese patients with OPLL [29]. Thus, possible genetic relationships among DISH, OPLL, and OLF remain to be elucidated.

Management

The management of thoracic myelopathy due to OPLL and OLF is challenging. First, the OLF makes posterior decompression necessary, but decompressive laminectomy in the thoracic spine is not very effective because the natural kyphosis restricts a backward shift of the spinal cord [30]. Hioki et al. [31] described a patient who underwent laminoplasty at C3–T1 and laminectomy at T2–3 due to OPLL at C3–T2 and OLF at T2–3. Postoperatively, the symptoms immediately and dramatically improved but then recurred at day 2, requiring an anterior decompression and fusion via a manubrium splitting approach. Second, if extirpation of OPLL is conducted, the spinal cord at the site of compression is vulnerable to damage. Several procedures for thoracic OPLL, including anterior, posterior, and combined anterior and posterior approaches, have been shown to have a high risk of post-operative neurological deterioration; the reported rates range from 2.7 to 18.8% [30, 32–35]. Tomita et al. [32] evaluated the outcomes of circumspinal decompression in the treatment of myelopathy due to OPLL and OLF. Eight cases underwent surgery by thoracotomy at the middle or lower thoracic spine and two cases by costotransversectomy at the upper thoracic spine. JOA scores improved postoperatively in nine patients, while neurological deficit became worse in one patient. Third, leakage of CSF often occurs during extirpation of the ossified ligaments, which often are strongly adhered to the dura mater. Takahata et al. [36] reported that leakage caused by a dural tear occurred in 12 of 30 patients (40%) who underwent circumferential spinal cord decompression via a single posterior approach [36]. In another study, complete resection of the OPLL caused leakage of CSF in 8 of 16 (50%) patients [30]. In two patients who underwent anterior decompression through thoracotomy, CSF leaked into the intrathoracic space, causing a hydrothorax that was very difficult to regulate [30]. Additionally, massive postoperative epidural venous bleeding, which required a second operation, and cystic swelling of the subarachnoid space were also reported with the anterior approach [33].

To evaluate the outcomes of different surgical methods in the treatment of thoracic OPLL, Matsumoto et al. [37] conducted a retrospective multi-institutional study in 2008. Despite the lack of statistically significant differences in the surgical outcomes among different surgical methods, neurological deterioration was observed more frequently in surgical procedures in which the OPLL was excised or thinned and floated than in those in which the lesion was left untouched. These authors concluded that dorsal shift and decompression of the spinal cord at the cervicothoracic junction could be expected when posterior decompression was used alone because the spinal curvature was usually

lordotic or only slightly kyphotic [37]. Given the above-mentioned factors and the characteristics of our patient, we decided to perform posterior laminectomy and resection of OLF without touching the anterior compressive OPLL.

Some researchers have recommended internal fixation after decompression to stabilize the spine, which could facilitate the recovery of the damaged spinal cord despite the anterior impingement of the spinal cord by the remaining OPLL [30]. In our case, we did not apply internal fixation for two reasons. First, in the upper thoracic spine, the sternal–rib complex, which was confirmed as the fourth column of the spine, could stabilize the spine [38]. Second, the anterior column had been fused by ossified ligaments in our patient, which provided sufficient stabilization. Postoperatively, the patient's symptoms were greatly improved and the JOA score increased from 5 preoperatively to 9 at final follow-up, which confirmed the effectiveness of posterior decompression.

Despite these results, our study did contain some defects. Although the patient was satisfied with the results and did not want to receive further anterior decompression, he experienced residual hyperreflexia and muscle strength in the lower extremities remained Grade 4/5. These physical signs might be due to the long-term anterior compression of OPLL against the spinal cord and the limited backward drift of spinal cord due to thoracic kyphosis. Leakage of CSF was confirmed at postoperative day 27 in our patient. This might have been caused by unrecognized damage to the spinal dura mater during decompression and by postoperative damage to the vulnerable spinal pia mater due to friction against rough and ossified ligaments.

Conclusions

This study reveals that upper thoracic OPLL and OLF can co-exist with DISH, causing compressive myelopathy. Further research on the relationships among DISH, OPLL, and OLF are required. The surgical procedure for thoracic OPLL and OLF is technically demanding.

Conflict of interest None of the authors has any potential conflict of interest.

References

- Matge G (2005) Surgical management of cervical radiculopathy in Forestier's disease. Case report and review. Neurochirurgie 51:15–18
- Ozkalkanli MY, Katircioglu K, Ozkalkanli DT, Savaci S (2006) Airway management of a patient with Forestier's disease. J Anesth 20:304–306. doi:[10.1007/s00540-006-0418-5](https://doi.org/10.1007/s00540-006-0418-5)
- McCafferty RR, Harrison MJ, Tamas LB, Larkins MV (1995) Ossification of the anterior longitudinal ligament and Forestier's disease: an analysis of seven cases. J Neurosurg 83:13–17. doi:[10.3171/jns.1995.83.1.0013](https://doi.org/10.3171/jns.1995.83.1.0013)
- Pappone N, Di Girolamo C, Del Puente A, Scarpa R, Oriente P (1996) Diffuse idiopathic skeletal hyperostosis (DISH): a retrospective analysis. Clin Rheumatol 15:121–124
- Chacko AG, Daniel RT (2007) Multilevel cervical oblique corpectomy in the treatment of ossified posterior longitudinal ligament in the presence of ossified anterior longitudinal ligament. Spine (Phila Pa 1976) 32:E575–E580
- Jun BY, Yoon KJ, Crockard A (2002) Retro-odontoid pseudotumor in diffuse idiopathic skeletal hyperostosis. Spine (Phila Pa 1976) 27:E266–E270
- Storch MJ, Hubbe U, Glocker FX (2008) Cervical myelopathy caused by soft-tissue mass in diffuse idiopathic skeletal hyperostosis. Eur Spine J 17(Suppl 2):S243–S247. doi:[10.1007/s00586-007-0508-6](https://doi.org/10.1007/s00586-007-0508-6)
- Patel NP, Wright NM, Choi WW, McBride DQ, Johnson JP (2002) Forestier disease associated with a retroodontoid mass causing cervicomедullary compression. J Neurosurg 96:190–196
- Goto S, Tanno T, Moriya H (1995) Cervical myelopathy caused by pseudoarthrosis between the atlas and axis associated with diffuse idiopathic skeletal hyperostosis. Spine (Phila Pa 1976) 20:2572–2575
- Pascal-Moussellard H, Drossard G, Cursolles JC, Catonne Y, Smadja D (2006) Myelopathy by lesions of the craniocervical junction in a patient with forestier disease. Spine (Phila Pa 1976) 31:E557–E560
- Griffiths ID, Fitzjohn TP (1987) Cervical myelopathy, ossification of the posterior longitudinal ligament, and diffuse idiopathic skeletal hyperostosis: problems in investigation. Ann Rheum Dis 46:166–168
- Pouchot J, Watts CS, Esdale JM, Hill RO (1987) Sudden quadriplegia complicating ossification of the posterior longitudinal ligament and diffuse idiopathic skeletal hyperostosis. Arthritis Rheum 30:1069–1072
- Chiba H, Annen S, Shimada T, Imura S (1992) Atlantoaxial subluxation complicated by diffuse idiopathic skeletal hyperostosis. A case report. Spine (Phila Pa 1976) 17:1414–1417
- Kawabori M, Hida K, Akino M, Yano S, Saito H, Iwasaki Y (2009) Cervical myelopathy by C1 posterior tubercle impingement in a patient with DISH. Spine (Phila Pa 1976) 34:E709–E711
- Epstein NE (2000) Simultaneous cervical diffuse idiopathic skeletal hyperostosis and ossification of the posterior longitudinal ligament resulting in dysphagia or myelopathy in two geriatric North Americans. Surg Neurol 53:427–431 (discussion 431)
- Razmi R, Khong KS (2001) Cervical cord injury in an elderly man with a fused spine—a case report. Singap Med J 42:477–481
- Alenghat JP, Hallett M, Kido DK (1982) Spinal cord compression in diffuse idiopathic skeletal hyperostosis. Radiology 142: 119–120
- Reisner A, Stiles RG, Tindall SC (1990) Diffuse idiopathic skeletal hyperostosis causing acute thoracic myelopathy: a case report and discussion. Neurosurgery 26:507–511
- Wilson FM, Jaspal T (1990) Thoracic spinal cord compression caused by diffuse idiopathic skeletal hyperostosis (DISH). Clin Radiol 42:133–135
- Chi D, Miyamoto K, Hosoe H, Kawai G, Ohnishi K, Suzuki N, Sumi H, Shimizu K (2008) Symptomatic lumbar mobile segment with spinal canal stenosis in a fused spine associated with diffused idiopathic skeletal hyperostosis. Spine J 8:1019–1023
- Utsinger PD (1985) Diffuse idiopathic skeletal hyperostosis. Clin Rheum Dis 11:325–351
- Crockard HA, Sett P, Geddes JF, Stevens JM, Kendall BE, Pringle JA (1991) Damaged ligaments at the craniocervical junction presenting as an extradural tumour: a differential diagnosis in the elderly. J Neurol Neurosurg Psychiatry 54:817–821

23. Ono M, Russell WJ, Kudo S, Kuroiwa Y, Takamori M, Motomura S, Murakami J (1982) Ossification of the thoracic posterior longitudinal ligament in a fixed population. Radiological and neurological manifestations. *Radiology* 143:469–474
24. Ehara S, Shimamura T, Nakamura R, Yamazaki K (1998) Paravertebral ligamentous ossification: DISH, OPLL and OLF. *Eur J Radiol* 27:196–205
25. Resnick D, Guerra J Jr, Robinson CA, Vint VC (1978) Association of diffuse idiopathic skeletal hyperostosis (DISH) and calcification and ossification of the posterior longitudinal ligament. *AJR Am J Roentgenol* 131:1049–1053
26. Hukuda S, Mochizuki T, Ogata M, Shichikawa K (1983) The pattern of spinal and extraspinal hyperostosis in patients with ossification of the posterior longitudinal ligament and the ligamentum flavum causing myelopathy. *Skeletal Radiol* 10:79–85
27. Wiberg C, Klatt AR, Wagener R, Paulsson M, Bateman JF, Heinegard D, Morgelin M (2003) Complexes of matrilin-1 and biglycan or decorin connect collagen VI microfibrils to both collagen II and aggrecan. *J Biol Chem* 278:37698–37704
28. Tsukahara S, Miyazawa N, Akagawa H, Forejtova S, Pavelka K, Tanaka T, Toh S, Tajima A, Akiyama I, Inoue I (2005) COL6A1, the candidate gene for ossification of the posterior longitudinal ligament, is associated with diffuse idiopathic skeletal hyperostosis in Japanese. *Spine (Phila Pa 1976)* 30:2321–2324
29. Havelka S, Vesela M, Pavelkova A, Ruzickova S, Koga H, Maeda S, Inoue I, Halman L (2001) Are DISH and OPLL genetically related? *Ann Rheum Dis* 60:902–903
30. Yamazaki M, Mochizuki M, Ikeda Y, Sodeyama T, Okawa A, Koda M, Moriya H (2006) Clinical results of surgery for thoracic myelopathy caused by ossification of the posterior longitudinal ligament: operative indication of posterior decompression with instrumented fusion. *Spine (Phila Pa 1976)* 31:1452–1460
31. Hioki A, Miyamoto K, Hosoe H, Shimizu K (2008) Two-staged decompression for thoracic paraparesis due to the combined ossification of the posterior longitudinal ligament and the ligamentum flavum: a case report. *Arch Orthop Trauma Surg* 128:175–177. doi:[10.1007/s00402-007-0336-5](https://doi.org/10.1007/s00402-007-0336-5)
32. Tomita K, Kawahara N, Baba H, Kikuchi Y, Nishimura H (1990) Circumspinal decompression for thoracic myelopathy due to combined ossification of the posterior longitudinal ligament and ligamentum flavum. *Spine (Phila Pa 1976)* 15:1114–1120
33. Hanai K, Ogikubo O, Miyashita T (2002) Anterior decompression for myelopathy resulting from thoracic ossification of the posterior longitudinal ligament. *Spine (Phila Pa 1976)* 27:1070–1076
34. Fujimura Y, Nishi Y, Nakamura M, Toyama Y, Suzuki N (1997) Long-term follow-up study of anterior decompression and fusion for thoracic myelopathy resulting from ossification of the posterior longitudinal ligament. *Spine (Phila Pa 1976)* 22:305–311
35. Kurosa Y, Yamaura I, Nakai O, Shinomiya K (1996) Selecting a surgical method for thoracic myelopathy caused by ossification of the posterior longitudinal ligament. *Spine (Phila Pa 1976)* 21:1458–1466
36. Takahata M, Ito M, Abumi K, Kotani Y, Sudo H, Minami A (2008) Clinical results and complications of circumferential spinal cord decompression through a single posterior approach for thoracic myelopathy caused by ossification of posterior longitudinal ligament. *Spine (Phila Pa 1976)* 33:1199–1208
37. Matsumoto M, Chiba K, Toyama Y, Takeshita K, Seichi A, Nakamura K, Arimizu J, Fujibayashi S, Hirabayashi S, Hirano T, Iwasaki M, Kaneoka K, Kawaguchi Y, Ijiri K, Maeda T, Matsuyama Y, Mikami Y, Murakami H, Nagashima H, Nagata K, Nakahara S, Nohara Y, Oka S, Sakamoto K, Saruhashi Y, Sasao Y, Shimizu K, Taguchi T, Takahashi M, Tanaka Y, Tani T, Tokuhashi Y, Uchida K, Yamamoto K, Yamazaki M, Yokoyama T, Yoshida M, Nishiwaki Y (2008) Surgical results and related factors for ossification of posterior longitudinal ligament of the thoracic spine: a multi-institutional retrospective study. *Spine (Phila Pa 1976)* 33:1034–1041
38. Shen FH, Samartzis D (2007) Successful nonoperative treatment of a three-column thoracic fracture in a patient with ankylosing spondylitis: existence and clinical significance of the fourth column of the spine. *Spine (Phila Pa 1976)* 32:E423–E427